A CASE OF TUBERCULAR MENINGITIS, WITH AUTOPSY.[1]

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I DESIRE to present for the consideration of the Society the following history, with the brain, of a patient who died of tubercular meningitis on the 20th of September last. The symptomatic observations made during the progress of the case are almost absolutely accurate, even to the minutest details, a fact which should add materially to the value and interest of the history. Records were made for ten days previous to death, of temperature, pulse, and respiration, at three-hour intervals and the nurse, having been thoroughly posted in advance, was constantly on the *qui vive* for any symptoms which were to have been expected. A summary of the salient points in the history is as follows:

The patient, a Scotchman by birth, thirty-eight years old, was a man of delicate physique, small in stature, and weighing about 110 pounds. In temperament he was typically Scotch, reticent, nervously sensitive, and with great tenacity of purpose. Thoroughly educated in his youth, he had continued all his life a student. By occupation he was an expert designer and manager for a large lithographing establishment in this city. During the past fifteen years he had suffered from four or more pulmonary hemorrhages, the last about fifteen months ago. He had never had syphilis. At the time of the occurrence of the last hemorrhage he consulted Dr. Loomis, Sr., and was advised to go to Colorado at once, his case being pronounced one of phthisis. To leave New York at that time meant the overthrow of cherished business plans and great financial loss. The other alternative was not more reassuring. The dilemma proved a source of great mental worry and anxiety, which, with extraordinary, though largely assumed responsibility in business and close application to work, brought on a condition of apparent cerebral neurasthenia. Thirteen months ago he began to be troubled with headaches and insomnia, both of which gradually increased in degree. During the past five months his wife had been absent in Europe, awaiting the results of her first confinement, though married eight years. Anxiety as to the outcome, his wife being far distant in case of danger, was a factor largely increasing his mental worry, and a stranger in a strange land, having no confidants, he was forced to a dangerous self-communion.

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[1] Presented, with specimen, to New York Neurological Society, Oct. 7th, 1890.

At the time of my first visit, September 2d, I found the patient sitting in a darkened room, with the attitude and expression of utter mental exhaustion. The only symptoms complained of, in addition to the two mentioned above, were a slight nausea (no vomiting), a feeling of intense prostration and constipation. He stated that three hours in the twenty-four represented the maximum of sleep obtained for several weeks previously. The headache or pains he located in the region of the occiput, the upper neck and the brow. There was no corresponding pain on pressure or percussion in either region. This one symptom (headache) almost entirely monopolized the patient's attention. Physical examination showed an area of disease in the right lung at apex, an enlarged liver, a coated tongue, and nothing more. He had not had any cough, night sweats, or fever, and his weight had remained stationary for a year. The pulmonary trouble had been, therefore, for some time inactive. The temperature, pulse, and respiration were normal. A tentative diagnosis of cerebral neurasthenia was made, I being unable, at that time, to decide that the symptoms were due to organic disease. Two days later I saw the patient again, this time at my office, he having so far improved under symptomatic treatment as to have returned to his duties, I made an application of galvanism to the brain at this time (1.5 miliampères for three minutes) with decided, though temporary, relief of the headache. Although the patient was directed to call again on Sunday (September 7th), he did not do so. When reminded of his engagement with the doctor by one of his household, he remarked: "What doctor?" "Dr. Pritchard," was the reply. "Who is Dr. P.?" he asked. His temporary confusion of mind clearing up in a few moments however, he requested that I be sent for and greeted me with his usual courtesy, and in an hour's conversation subsequently, upon various subjects, he showed a degree of brilliancy in his conception of ideas and of logical accuracy in argument which was beyond cavil. Upon being questioned he stated that he had noticed transient conditions of mental confusion, with inability to systematize his ideas and a limitation of his vocabulary. His manner, unless specially aroused by some unusual mental stimulus, was decidedly apathetic and melancholy. He sat in a darkened room by preference, although there was no decided photophobia. His temperature by mouth was still normal; pulse 72. The patient's condition remained practically unchanged up to Wednesday, September 10th, when, for the first time, the temperature rose above normal, the thermometer registering $101.\frac{4}{10}$, pulse 76. Vomiting also occurred for the first time, the emesis being slight and consisting apparently of a simple regurgitation. It did not recur. The patient's mental state was one of increased confusion and apathy, possibly due in part to the action of thirty grains each of sodii bromid. and sulfonal, with seven and a half grains phenacetine, taken in two doses the previous evening, the result being ten hours sleep and a diminution of the headache. The diagnosis of tubercular meningitis, already suspected, was no longer considered a matter of doubt, and a prognosis given accordingly.

I shall confine my report of the subsequent clinical aspects of the case to a brief resume of the more essential particulars. The highest temperature observed, from the date of onset of febrile symptoms up to within twelve hours of patient's death, was 102.8 on the third and fourth days; the lowest 99.8 on the sixth day. The average temperature, up to within twelve hours of death, taken at three-hour intervals, was 101.4. Respiration varied from 26 to 56, and was almost entirely abdominal for twenty-four hours before death. The pulse was typical, ranging from 60 to 70 until towards the close, when it ran quickly to 150, 160, and beyond. The first symptom indicating a paretic state was constipation, which persisted for a week in spite of varied and persistent efforts to overcome it, elaterium, with massage of the abdomen for an hour, finally producing the desired effect. A mild degree of right ptosis appeared on the fourth day and remained until within forty-eight hours of death, when it disappeared. It was at no time marked. A loss of the light reflex, with immobility of the pupil, was noticed about the same time; but no strabismus appeared during the entire progress of the case.[1] Some stiffness of the neck and a tendency to burrow in the pillows appeared early, but there was never a condition approaching opisthotonos. On Saturday, the fourth day of temperature, speech became thick, much less intelligible, though deglution was not then interfered with, the patient taking and retaining large quantities of liquid nourishment. Mental hebetude had given place to restless, low delirium, with subsultus tendinum and occasional risus sardonicus. The patient was seen at nine P. M. on this day, by Dr. Landon Carter Gray, in consultation with the writer, and the diagnosis of tubercular meningitis confirmed. Slight epistaxis occurred on the fifth day, and the patient passed urine involuntarily for the first time.

The absence of cremasteric and abdominal reflexes was noticed on the same day, as well as the presence of taches cerebrale. From this time the urine was passed through catheter, the quantity being enormous, and sugar being present by analysis. The condition of the patient remained about the same until the evening of Sept. 17th, three days before death, when speech became almost unintelligible, hebetude or stupor greater, and deglutition very difficult and imperfect. The pulse ran up from 80 to 140 in a few hours, respiration becoming also affected. Considering these symptoms an indication of serious involvement of the vagus, death was hourly expected. All food and medicine had to be discontinued by mouth. To combat the impending failure of heart and respiration, $^1/_{25}$ gr. strychnine was administered hypodermically, and repeated in three hours. These injections were continued at irregular intervals, and with decided effect in stimulating both respiration and pulse. Two days before death it was noticed that the patient, in his restless and ataxic movements of the extremities, scarcely used the right leg or arm at all, and this condition gradually developed into a distinct right hemiparesis.

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[1] Ophthalmoscopic examination of the fundus revealed negative results.

On the morning of the 19th there occurred a discharge of thick muco-purulent offensive matter from the nostrils, which continued up to within an hour or two of death. It escaped with each expiratory movement, and the amount was estimated to have been at least four or five ounces. Toward the close the temperature and pulse rose rapidly—the former registering, half hour before the end, 106° in the axilla. Death occurred quietly. There was no convulsion at any time, nor was there the slightest tendency manifested in that direction.

I fortunately secured an autopsy, which was made, ten hours after death, by my friend Dr. E. G. Mason, assisted by the writer. The contents of the skull alone were examined, in accordance with a promise previously exacted. Upon removing the scalp, a cup-shaped excavation in the centre of the external surface of the right parietal bone was observed. It was about as large in circumference as a silver dime, and was the result of a local necrotic process, probably tubercular. It did not extend to the inner table, and there was not the slightest pathological change in the corresponding subjacent meninges. There was also a depressed linear fracture (quite old) of the outer table of the right frontal bone, about half an inch above the orbital ridge. Although at first there appeared to be some atrophy of the convolutions corresponding in location to this fracture, it was afterward determined that this appearance was the result of a temporary derangement in handling of the hemispheres. The bones of the skull were unusually thin. Superficially examined before removal, the dura was seen to be studded at irregular intervals, along the convexity of both hemispheres, with granular masses, the surrounding meningeal tissue being intensely congested for a limited area. These small masses, upon section, were cheesy in consistency, and were evidently pathological in character. They were differentiated from the Pachionian bodies, and were presumably tubercular. Upon removal, the brain (which I show you to-night) was found to be normal in size and in the development of the convolutions. It weighed fifty ounces. The dura was adherent only at two or three places, principally at the base, where the essential pathological conditions were found, so far as the pathology of the case has been investigated. It will have been noticed that the specimen presented is that of a perfectly intact brain, no sections having been made. This was unavoidable in order to show the characteristic appearance of the basal ganglia, which would have been impossible had the brain been dissected. The condition observed at the base of the brain, upon removal, corresponded to a lepto-meningitis involving the upper medulla and pons, the crura cerebri, and all the cranial nerves perhaps. The location of the area of disease had been easily determined by the symptoms observed during life, and the correspondence was exact in location, but by no means in degree. The area described was found covered with purulent exudation, perhaps two ounces in amount. This was of viscous consistency, and clung tenaciously to the surface of the brain. The brain-tissue itself was in a state of beginning disintegration or necrosis, and the sheaths of the nerve-trunks were filled with pus. The pia-arachnoid was intensely injected in places and in others softened and easily torn. No tubercles, clearly demonstrable as such, could be found in the pia-arachnoid at any point.

The case, to summarize it briefly, offers more than one inviting field for speculation and inquiry. Considering it first from an etiological standpoint, it would be interesting to know the exact relationship of the patient's temperament, his mental surroundings, and accumulated sources of anxiety and brain worry, to the development of tubercles in the meninges. What relationship, if any, existed between the apparently stationary condition of the pulmonary disease to its cerebral development? In other words, are we to expect, in patients affected with tuberculosis of a similar temperament and surroundings, the development of the cerebral disease, and cannot such development be prevented by a proper recognition of this fact, if fact it be, with the enforcement of brain rest? Coming next to the clinical aspects of the case, could an earlier diagnosis have been made? Obstinate insomnia and headaches, it will be remembered, were the only subjective symptoms manifested until within two weeks of a fatal termination. Would it not, however, have been perfectly justifiable to have diagnosed cerebral tuberculosis with these two symptoms alone, knowing that the patient suffered from the pulmonary disease? Viewing the case from a therapeutic standpoint, the "old-new" question suggests itself: Has the surgeon a place in the treatment of such cases? Here was pus, and a surgical axiom demands that pus be evacuated wherever found. The skull has been entered so often for this purpose that it is now an old story. If we are to consider the spontaneous escape of pus, which occurred in this case, a cue from nature, the question is already answered in the affirmative, and the surgeon has but to imitate.

The most interesting features of the whole case, however, are to be found in the results of the autopsy, the most remarkable being the gross disparity between the very grave and extensive lesions found post-mortem and the comparative mildness (although the patient died) of the corresponding paralytic symptoms observed during life. Six or eight ounces of offensive pus (including the amount discharged through the nares and the ounce or two found at the autopsy) would, one would think, have provoked symptoms of a much more pronounced character, involving by pressure, as it did, such an extensive area and structures of such vital importance and sensitiveness. The only solution of the enigma is to be found in the oft-repeated statement, that the brain, in spite of theories to the contrary, is capable of resisting, to an enormous and unknown degree, the encroachments of disease or injury of even the gravest character.